Air Quality Data and Patients with COPD: an information gap

A research proposal submitted to the EPA April 29, 2003 by F. Eugene Yates, M.D.,
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Chronic Obstructive Pulmonary Disease (COPD)

COPD is the fourth leading cause of death in the United States today (after heart disease, cancer and stroke). Over 16 million have COPD in the U.S., accounting for more than 16 million office visits, 500,000 hospitalizations, and at least $18 billion in direct health care costs (1). It may begin in early adult life, but is progressive, so that its manifestations are more likely to be seen in elderly people. Cigarette smoking has been the chief cause, but even in those who never smoked themselves it is appearing with increasing frequency. The provocative agents are in the air breathed, and may be particulates, gases, water vapor, or air temperature extremes (see below). The pathophysiology defining the disease consists of increased airway resistance during expiration (bronchial spasms), increased mucus (phlegm) production, wheezing and coughing. Lungs and bronchi are being damaged by macrophages and neutrophils – inflammatory cells with anti-elastin enzymes. The end result is bronchitis, loss of lung elasticity, decreased alveolar exchange surfaces in the lung, emphysema, increased work of breathing, oxygen starvation, carbon dioxide retention, acidosis, cor pulmonale (damage to the right ventricle of the heart), severe limitations on physical activity, shortness of breath, weight loss, recurrent infections, dependency, depression and death. It is a prolonged and unpleasant dying. Although COPD has some resemblance to asthma, it has a very different pathophysiological basis. It is not asthma., and does not have a primary allergic component. Instead the subjects have an abnormal inflammatory response to noxious particles or gases in the air (2), disturbing the airway mechanics (3).

A few cases have a genetic basis (α_1-antitrypsin deficiency), but this inherited form is uncommon.

Air quality causes provocative of acute attacks of respiratory distress in subjects with COPD

Some Industrial causes (particles)

Well-recognized industrial threats to air quality include: 1) smoke (firefighters), 2) beryllium, 3) asbestos, 4) coal dust, 5) silica dust, 6) emissions from coal-burning power plants.

Some non-industrial causes (particles and gases)

Cigarette smoke (primary or secondary), wood-smoke, mites, fungi, house-dust, low-humidy dry air, cold air, car and truck exhausts. Carbon monoxide is not itself

provocative, but in people with compromised lung function and deficient oxygenation, it becomes an enhanced threat. However, thanks to the efforts of the EPA, carbon monoxide is no longer a major risk factor with respect to air quality (except in a few spots, like Calexico, CA, and Lynwood, CA just south of Los Angeles, where local air flow patterns impose the brunt of carbon monoxide emissions from Mexico or greater Los Angeles.)

Medical paradox in treating COPD – exercising in "bad air"

Though it seems counter-intuitive, a major component of therapy for COPD is exercise (to strengthen respiratory muscles and increase endurance). The recommended schedule is 30 minutes of "aerobic" exercise, 4-7 times per week. A question immediately arises: *"Doesn't exercise increase respiration and exposure to provocative agents in the inspired air, thereby further damaging the lungs?"*

It is evident that patients with COPD should not exercise during times or in places having "bad air". But how are they to know? Yes, air quality information is available in a general way from EPA (especially about outside air) and sometimes reported in the local news, and from OSHA (about inside air in public buildings, and factories, etc.) but neither physicians nor patients know where to get the information, or what measurements lie behind these assessments, and, even if they did know, there would still be the uncertainty about which components of "bad air" were actually relevant to a particular patient's disease.

There is a research and information gap to be filled.

RESEARCH PROPOSAL FOR EPA (OUTLINE ONLY)

Summary

A group of patients (aged 50 years or older, men and women) with diagnosed COPD, and living in a city with known variability in air quality (e.g., Los Angeles) are to be provided (by the EPA) with (inexpensive but effective) peak airflow meters. (See Figure 1 for example.) These are small, easy to use, and within a few seconds provide a quantitative estimate of the instantaneous condition of the airway resistances. These measurements are to be made three times a day, morning noon and night, always at the same times, and recorded on a data sheet provided by the EPA (see Figure 2 for example). At the time of a measurement the patient is not to have just used a bronchodilator, and is not to have just finished exercising. Measurements are to be made while standing at rest.

Each month the filled data sheets are to be forwarded to a designated EPA site. The patient information about variations in disease status will then be correlated with data the EPA has collected concerning their view of "air quality" at the same times. The question to be answered is:

"For this patient, which, if any, component of the EPA scoring of air quality, regularly correlates strongly with acute decreases in peak flow (respiratory function)?"

Use of the whole data set from all subjects

The complete set will reveal whether or not there is any relevance at all for patients with COPD to be found in the EPA assessments of air quality.

The data set will also answer whether there is a general correlation between aspects of estimated air quality that is relevant for most or all patients, or whether each patient tends to have his/her own vulnerabilities to air pollution or humidity/temperature profiles.

Predictions

1. I expect that there will be a statistically significant correlation between *some* component(s) of the EPA estimates of air quality and the clinical status (peak airflow estimates) of patients.

2. I expect that a few of these relevant components will apply *in general* to the population with COPD.

3. I expect that there will be a time-of-day pattern (as is well known already) between air quality as seen by the EPA and in peak airflow measurements as seen by patients.

4. I expect that there will be a day-to-day variation in the data (both EPA data and patient data) at any given time of day.

If these expectations are realized, we will have a basis for producing a medically-relevant air quality bulletin, for physicians and patients, that will make clear exactly when is the optimum time for exercise (indoors or outside) for patients in this locale, with COPD, and when exercise should be avoided.

NOTE: Though my suggested study is for the benefit of patients with COPD, and their physicians, it is obvious that ALL people exercising will benefit!

REFERENCES

There is an extensive medical literature on COPD and many excellent reports from EPA and OSHA on air quality. Here I list just a few indicative references. (More are available on request.)

1. Snow, V. et al. (2001) For the Joint Expert Panel on Obstructive Pulmonary Disease of the American College of Chest Physicians and the American College of Physicians-American Society of Internal Medicine. Evidence base for management of acute exacerbations of chronic obstructive pulmonary disease. Ann Internal med. 134: 595-599.
2. Pauwels, RA et al. (2001) Global strategy for the diagnosis, management and prevention of chronic obstructive pulmonary disease. NHBLI/WHO Global Initiative for Chronic Obstructive Lung Disease (GOLD)., Executive summary. Resp. Care 46: 798-825. (see also www.gpld-copd.com)
3. Kamm, R.D. (1996). Small airway wall mechanics: an integrative approach. BMES Bulletin 20: 51- 55.

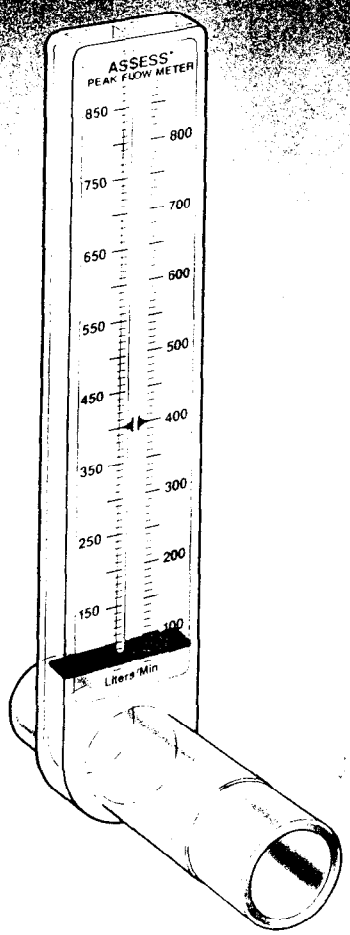
FULL RANGE

ASSESS®

Peak Flow Meter

Instructions For Use

Recorder No. 7[illegible]



HEALTHSCAN
PRODUCTS INC. a division of
HEALTHDYNE TECHNOLOGIES

Peak Flow Meter

POCKETPEAK

Universal Range • 50-720 LPM
Catalog Number: 1801

Contents:
One Meter
Instructions For Use
Daily Record Chart
Zone Labels

Distributed By:

HUDSON RCI

FIG 1 TWO MODELS

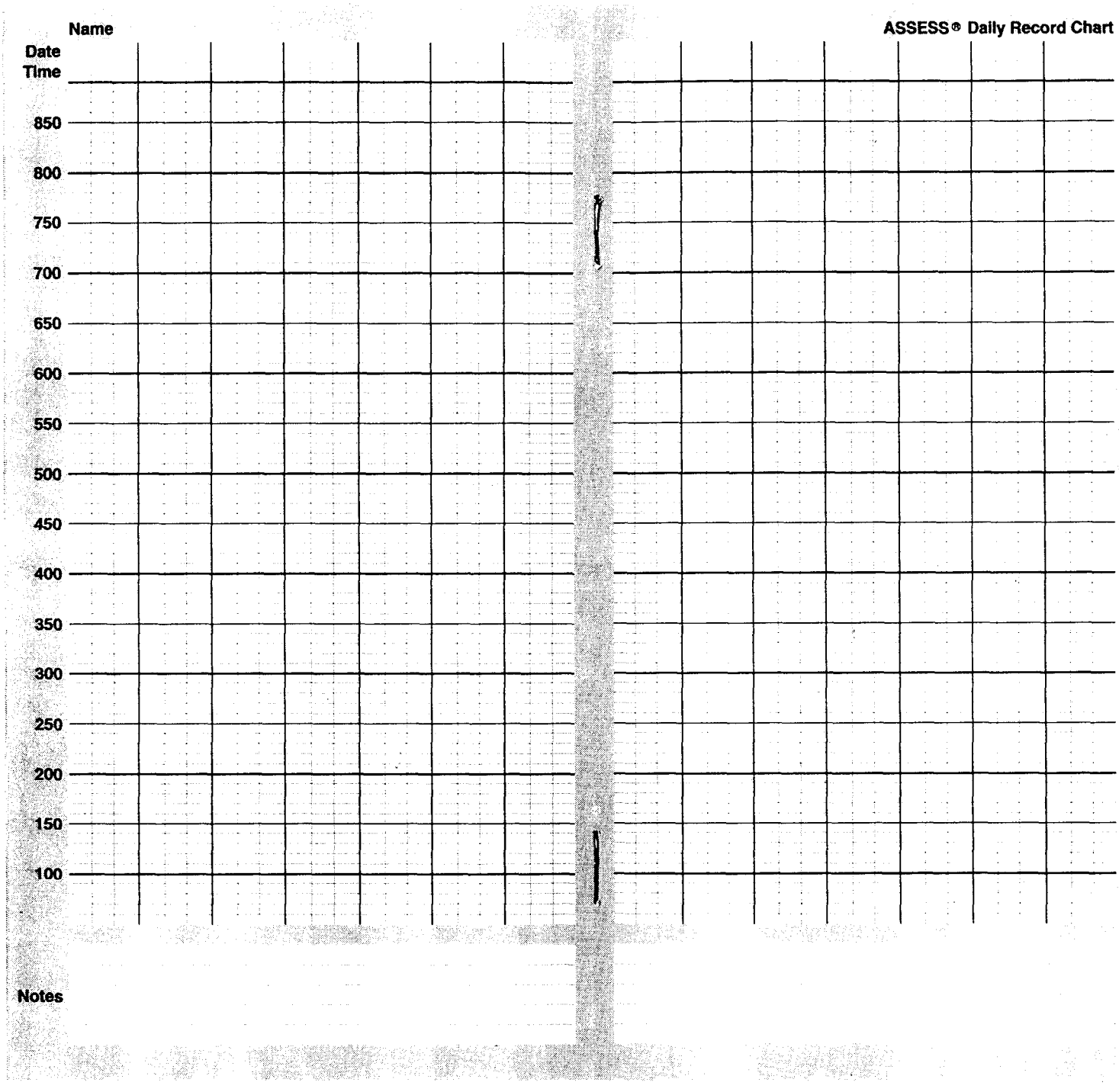
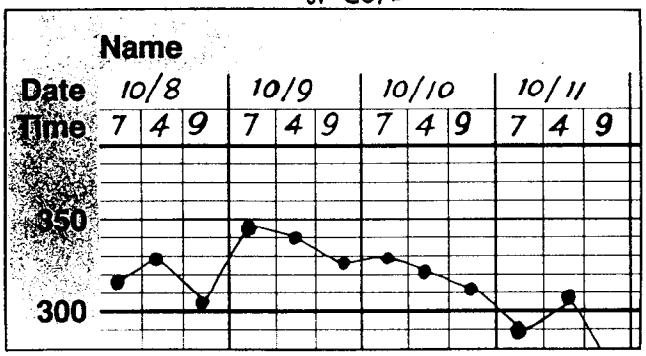


FIG 2A. PATIENT DATA SHEET

YOUR PEAK FLOW READINGS

- Consult your doctor to ensure you are recording your values correctly.
- Make sure that you keep your records up-to-date.
- Your doctor will provide you with a management plan based on your peak flow measurements. Contact your doctor when changes in readings occur that may indicate worsening of your condition, as per his or her instructions.
- Taking daily peak flow readings will help your doctor make important decisions about your asthma treatment.

or COPD



FOR MULTIPLE PATIENT USE PLEASE NOTE

As a reminder to healthcare providers, note that when a single ASSESS unit is used repeatedly as a screening device, patients should be instructed to inhale *before* placing the instrument to their mouths.

Always use *disposable* mouthpieces for this application (Safety One-Way Valve Disposable Mouthpieces, Reorder No. 713; Pediatric Disposable Mouthpieces, Reorder No. 712; Adult Disposable Mouthpieces, Reorder No. 711).

The instrument may be cleaned and disinfected by most cold sterilization methods; e.g., Cidex® and Cidex 7® Activated Dialdehyde Solution (Note: Cidex Plus® should *not* be used).* After cleaning with these products, flush thoroughly with warm water from the mouthpiece end of the meter, in order to rinse the piston spring of any cleaner residue.

The meter may be exposed to ethylene oxide (EtO) sterilization methods (cycle temperatures not to exceed 120°F).

Never boil or autoclave the instrument.

*Cidex®, Cidex 7® and Cidex Plus® are trademarks of Johnson & Johnson Medical Inc., Arlington, Texas.

NORMAL PREDICTED AVERAGE PEAK EXPIRATORY FLOW (liters per minute)

The National Asthma Education and Prevention Program recommends that a patient's "personal best" be used as his/her baseline peak flow. "Personal best" is the maximum peak flow rate that the patient can obtain when his/her asthma is stable or under control. The following tables are intended as guidelines only.

NORMAL MALES*

Age (Years)	Height (in) 60" (cm) 152	65" 165	70" 178	75" 191	80" 203
20	554	575	594	611	626
25	580	603	622	640	656
30	594	617	637	655	672
35	599	622	643	661	677
40	597	620	641	659	675
45	591	613	633	651	668
50	580	602	622	640	656
55	566	588	608	625	640
60	551	572	591	607	622
65	533	554	572	588	603
70	515	535	552	568	582
75	496	515	532	547	560

or COPD

NORMAL FEMALES*

Age (Years)	Height (in) 55" (cm) 140	60" 152	65" 165	70" 178	75" 191
20	444	460	474	486	497
25	455	471	485	497	509
30	458	475	489	502	513
35	458	474	488	501	512
40	453	469	483	496	507
45	446	462	476	488	499
50	437	453	466	478	489
55	427	442	455	467	477
60	415	430	443	454	464
65	403	417	430	441	451
70	390	404	416	427	436
75	377	391	402	413	422

CHILDREN AND ADOLESCENTS†

Height (Inches)	Males & Females	Height (Inches)	Males & Females
43	147	55	307
44	160	56	320
45	173	57	334
46	187	58	347
47	200	59	360
48	214	60	373
49	227	61	387
50	240	62	400
51	254	63	413
52	267	64	427
53	280	65	440
54	293	66	454

*Nunn, AJ, Gregg I: *Brit Med J* 298:1068-70, 1989.

†Polgar G, Promadhat V: *Pulmonary function testing in children: Techniques and standards.* Philadelphia, W.B. Saunders Company, 1971.

NOTE: All tables are averages and are based on tests with a large number of people. The peak ow rate of an individual can vary widely. Individuals at altitudes above sea level should be aware that peak flow readings may be lower than those at sea level, which are provi d in the tables.

FIG 2B. - REFERENCE VALUES